





# REMARKS ON THE HEART IN DEBILITY.

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SEVERAL years ago considerable interest was shown in certain discussions as to the cause of the clinical facts observed in cases of cardiac debility. The principal subjects under debate at that time were the explanations which had been advanced regarding two of the phenomena commonly observed in feeble conditions of the heart. The first of these, and that the more frequent in its occurrence, is the systolic murmur heard in the second left intercostal space, at or near the pulmonary area; the second, not so often presenting itself to the observer, is the systolic impulse seen and felt in the same locality. To the investigation and explanation of these appearances several observers devoted much attention, and, inasmuch as many of the points connected with the physical signs under discussion were virtually settled, it may seem unnecessary to bring the subject forward again. As one of those, however, who took part in the discussions on this question, it seems to me nothing more than simple justice to those whose views then differed from my own to state frankly and candidly the opinions which have been borne in upon me since the time referred to. In attempting to do so, it will be my endeavour to be as brief as possible, and, in particular, to avoid unnecessary reference to older observers. This may be done the more easily as William Russell has given a very complete and most masterly summary of the views of previous authors in his work on this subject.

For the present purpose it is only necessary to recall a few facts. In order to account for the systolic murmur and accompanying pulsation sometimes seen in the second left intercostal space, in cases of mitral incompetence, Naunyn advanced the hypothesis that both appearances are produced by the backward stream from the left ventricle into the left auricle. According to this view the systolic murmur is of mitral origin, and is conducted by the regurgitant current into the dilated left auricular appendix, while the pulsation is caused by the same stream distending the appendix and thereby producing an impulse

on the thoracic parietes. Balfour applied this hypothesis to the corresponding phenomena, so commonly seen in the feeble heart of anæmia and allied conditions. In his work on diseases of the heart, as well as in separate papers dealing with this special question, he has strongly advocated this explanation, and his opinions were warmly supported in some contributions made at the same time by myself. We were, however, unable to adduce any evidence obtained from morbid anatomy in favour of our views, and, although many of the clinical features appeared to be explained by them, they could not be regarded as resting on any sure pathological basis.

Russell brought to the elucidation of the questions under discussion a large number of clinical and pathological observations, from the consideration of which he came to very different conclusions. He showed that in many cases where the systolic pulsation in the second left intercostal space had existed before death, post-mortem examination proved that the impulse could only have been caused by the *conus arteriosus*, which, in consequence of dilatation of the right ventricle, was so far to the left as to occupy the site of pulsation in the left intercostal space between the second and third cartilages. With regard to the basic murmur, heard in cardiac debility, Russell proposed two explanations. He suggested that in some cases it might be produced by dilatation of the left auricle, which, pressing upwards upon the pulmonary artery, gives rise to a narrowing of its lumen, while in other cases it is simply the systolic murmur of tricuspid incompetence propagated upwards to the *conus arteriosus*.

The main points at issue in the discussions regarding this subject were very critically examined and judicially weighed by Bramwell in his systematic work. As the result of a very careful review of the arguments which have been advanced by Balfour and Russell, he rejects the theories of both with regard to the production of the basic systolic murmur, and attributes it to the sudden pulsation of a large blood wave of abnormal composition into the vessel, which he thinks may probably be dilated.

Handford holds that the pulmonary systolic murmur, which he describes as disappearing in the erect position and reappearing when the patient is recumbent, is produced by the pressure of an enlarged, flabby, and dilated heart on the pulmonary artery.

Foxwell has, like Russell, found the pulmonary artery to be displaced considerably upwards. He regards the murmur in the pulmonary area as caused by a complicated change in the shape and position of the pulmonary artery, whereby its curve becomes increased, its axis and that of the right ventricle lie at a different angle from that existing under healthy conditions, and the vessel is flattened against the aorta. At the same time, however, he accepts Russell's view of a distended right auricle as the cause of the murmur in some cases.

In the last place, Sansom, after an examination of the views of

Balfour and Russell, which leads him to dissent from both, advances the opinion that the basic murmur can be initiated at the over-strained portion of the right ventricle, the conus just below the pulmonary valves, by the production of a fibrillar tremor. He is, however, also inclined to believe that the cusps may themselves vibrate in the current.

It is easy for me now to consider the questions involved in a perfectly dispassionate and impartial spirit, inasmuch as Russell has, in my opinion, absolutely disproved the views of all observers previous to himself. He has demonstrated, beyond all possibility of doubt, that the left auricle never reaches the anterior wall of the thorax, and that the pulsation in the second left intercostal space is produced by the conus arteriosus. The observations of Foxwell, Harris, and Maekenzie support him in this, and it seems to me that the explanation of Naunyn and Balfour has been entirely refuted.

This decision leads of necessity to the further conclusion that the hypothesis of Naunyn and Balfour with regard to the origin of the systolic murmur heard in the pulmonary area falls to the ground, for since the left auricle never approaches the surface there is no medium for the conduction of a mitral murmur towards the base of the heart. But it must further be stated that, in a large proportion of cases, there is no evidence of any mitral incompetency, and that it is a mere begging of the question to assume it.

The view advanced by Bramwell may be regarded as in every respect a compromise, as Russell puts it, between the explanations of Hope and Beau, and it has, like their theories, been effectually disposed of by him.

But while granting freely that Russell has disproved all previous theories, it seems to me that part of his own explanation will not bear investigation. He has yet to prove that in early stages the left auricle is dilated. In truth, the conditions appear to be the very reverse of those which he postulates. The mitral cusps are very often perfectly competent, and as long as there is no mitral regurgitation the pressure in the pulmonary artery must be greater than that in the left auricle.

The explanation of Handford cannot be accepted, not only because the basic murmur is heard very frequently indeed while the patient is in the erect position, but also because it makes its appearance before there is any noteworthy enlargement of the ventricles. The same argument applies to the reasoning of Foxwell with equal cogency, while his experiment of forcing water into the right ventricle of a debilitated subject after tying the pulmonary artery is so unlike anything in nature that it cannot be held to prove anything.

Sansom, finally, is obviously in error, as, if over-strain of the ventricle were a valid cause for a murmur, such a phenomenon would be of much more common occurrence than is the case. Almost every case of chronic renal cirrhosis, for example, would be attended by a murmur produced by the strain thrown on the left ventricle.

In order to present the clinical features with which this paper is

concerned, in a concrete form, the following case is worthy of record. It has already been utilised for the illustration of another point in a paper recently published, which is in no way directly connected with the object of this contribution.

MAGGIE G., æt. 18, unmarried, engaged in household duties; was admitted to Ward 25 of the Royal Infirmary on 5th June 1893, complaining of pains in her wrists and elbows.

Her father and mother, both æt. 42, had always been in good health. She had four brothers and one sister, all very strong, but three brothers had died in infancy. The patient's social surroundings had always been good.

She had never been very robust, and four years before admission had suffered from a rheumatic attack, since when she had never felt very well. About four months before entering the Hospital, pains had begun in the joints and had persisted ever since.

On her admission the patient was found to be somewhat pale, with a bright spot on each cheek. The skin was moist. The temperature was normal. The tongue was slightly furred, but the digestive system was otherwise healthy. There was no symptom connected with the hæmopoietic system.

She complained of some palpitation and a slight degree of breathlessness. The pulse was of low tension and moderate volume, perfectly regular, and varying in rate from 80 to 90. There was some pulsation in the veins of the neck, and a very distinct impulse in the second left intercostal space. On palpation the apex beat was found to be in the fifth left intercostal space,  $3\frac{1}{4}$  in. from midsternum. The pulsation, systolic in time, in the second left intercostal space was found to be most distinct,  $1\frac{1}{4}$  in. from the midsternal line. A tracing obtained from it by means of a revolving cylinder is given in the

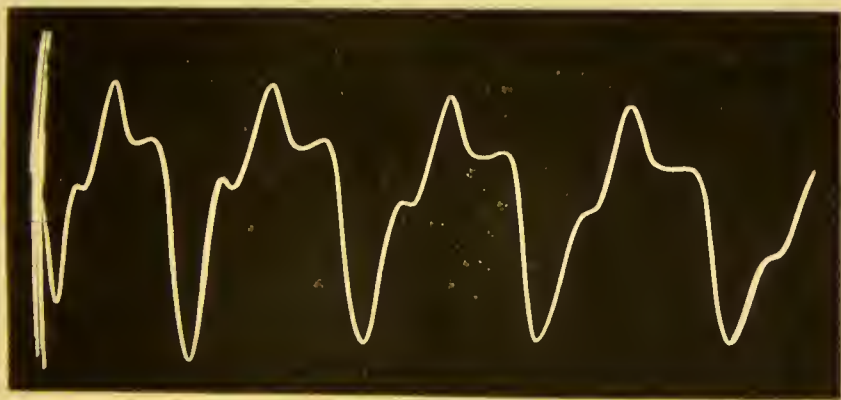


FIG. 1.

accompanying figure (Fig. 1). No thrill could be detected over any part of the præcordia. The cardiac dulness extended to 1 in. to the right and 4 in. to the left of the middle line at the level of the fourth rib. On auscultation, a venous hum was heard in the neck, and there were murmurs, systolic in rhythm, over the whole præcordia, which, on careful analysis, proved to be twofold. Around the region of the apex beat, and with its maximum loudness in the fourth interspace  $3\frac{1}{2}$  in. from midsternum, there was a harsh blowing systolic murmur, conducted as far as the edge of the sternum to the right, and beyond the anterior axillary line to the left. Over almost the entire sternal region there was a soft blowing systolic murmur, quite different in character from that heard at the apex. It had the same tone throughout the whole sternal region, but it seemed to have two points of maximum intensity—to be

more exact, it was loudest in the pulmonary region, exactly over the area of pulsation, from which point it waned in its intensity in every direction until near the lower end of the sternum, when it became louder, again culminating at the point where the left side of the sternum was joined by the sixth costal cartilage; but in this situation the murmur was not quite so loud as over the area of pulsation in the pulmonary region. The second sound was frequently reduplicated, and the later of the two second sounds, which could by auscultation be determined to be that due to the pulmonic cusps, was instantly followed by a short, sharp, high-pitched murmur, perfectly soft in character. This murmur was extremely restricted in its distribution, being only heard over a small triangular area  $2\frac{1}{4}$  in. in vertical and 2 in. in horizontal measurement, extending along the left border of the sternum, from the lower border of the third costal cartilage to the upper border of the fifth. This murmur was perfectly soft in character, and was absolutely unlike the obstructive diastolic murmur which is found in mitral stenosis. It could not be due to aortic

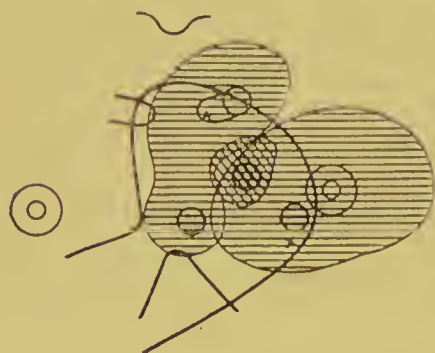


FIG. 2.

disease, of which there was no indication, and it could only, therefore, be a murmur of regurgitation, from the pulmonary artery into the right ventricle, due to the increased pressure and consequent dilatation of the orifice, with relative and transient incompetence of the cusps. The production of this murmur has been discussed by me in the paper referred to. All these murmurs are shown in Fig. 2, in which the areas over which the systolic murmurs were audible are marked by horizontal lines, while the diastolic murmur is shaded diagonally. The maximum intensities of the murmur are shown by the circles darkly shaded, and the impulses by the crosses.

The other systems presented no symptoms of disease, with the sole exception of a few crepitations at the bases of both lungs.

The diagnosis was cardiac dilatation, with mitral and tricuspid regurgitation, produced by the febrile affection, but it was considered probable that some stenosis of the mitral orifice might be insidiously progressing, although this was a mere supposition, not based on any direct evidence. The crepitations at the bases of the lungs were regarded as the expression of passive congestion from mitral incompetence, and the diastolic murmur was assumed to be one of pulmonary escape, in consequence of the strain on the artery from the high pressure within it.

By means of salol and similar remedies the patient was relieved of her rheumatic symptoms, and the administration of iron with other tonics greatly improved the cardiac condition. The diastolic murmur disappeared, and the lungs cleared up, but at the time of the patient's departure from the Royal Infirmary, on 17th July 1893, she still had the systolic murmurs, and the pulsation in the second intercostal space. She presented herself at the Hospital on the 2nd March 1894, when the diastolic murmur was found to be still absent, but the systolic murmurs were present as before. The first sound in the mitral area, preceding the systolic murmur, was, however, loud and elanging in

character, which seemed to support the view that a stenosis of the mitral orifice was gradually developing.

This case brings into prominence the systolic impulse in the second left intercostal space, as well as the systolic murmur in the same position. From the physical signs there could be no doubt of the presence of mitral and tricuspid incompetence, and it may be remarked here that the pulsation in the second left intercostal space is never observed except in cases which present so much dilatation as to allow of regurgitation at both auriculo-ventricular orifices. The diastolic murmur, which has been very fully discussed in the paper referred to, seems to me to be caused by regurgitation at the pulmonary orifice from high pressure and relative incompetence, and requires no further remark in this place. The systolic murmur heard over the sternal region of the præcordia appears to be the same throughout with two points of maximum intensity, and seems to me easily explained in this way, that, while at the lower end of the sternum it is heard with great distinctness, owing to the proximity of the muscular wall of the right ventricle and of the tricuspid valve, it is also heard with at least as much intensity over the conus arteriosus. It seems to me, in short, to be purely a murmur of regurgitation at the tricuspid orifice. While Russell's view with regard to the causation of the systolic pulsation in the second left intercostal space is to my mind absolutely proved, the murmur heard in that situation in the heart in debility seems to me to be simply a tricuspid systolic murmur propagated upwards by means of the conus arteriosus. It is quite analogous to the murmur produced at the right side of the heart in cases of heart strain, which is undeniably of tricuspid origin. To show that this murmur may have its greatest loudness close to the spot commonly known as the pulmonary area, the following case may be brought forward.

SYLVESTER N., æt. 24, unmarried, strapper in a stable; was admitted to Ward 6 of the Royal Infirmary, on 26th June 1893, with obvious symptoms of alcoholism.

His father, æt. 61, and mother, æt. 60, were in excellent health. Of ten brothers and sisters, only two brothers and one sister were alive, seven having died in their infancy. His social conditions were fairly good, except at times from his own fault.

The patient's previous health had been quite good, but he had been much addicted to drink.

The attack for which he was brought to the hospital began about Christmas, since which time he had been drinking very heavily, and about the middle of May pains in the legs, with some swelling of the ankles, set in.

On admission the patient was found to have great thirst and little appetite; the tongue was furred and shaky; the breath heavy and foul. No other symptoms connected with the alimentary or hæmopoietic systems were present.

There was breathlessness on exertion and swelling of the ankles and legs. The pulse was of low tension, moderate fulness, and perfect regularity. The rate was usually from 80 to 90. There was a well-marked venous pulsation in the neck. On inspection of the præcordia, no impulse could be seen, and the apex beat could only be felt when the patient was placed on his left side.

The deep cardiac dulness extended  $2\frac{3}{4}$  in. to the right, and  $4\frac{1}{2}$  in. to the left of the middle line at the level of the fourth cartilage. On auscultating the heart, a soft systolic murmur was heard over a great part of the præcordia, with its maximum intensity over the left half of the sternum opposite the attachment of the third cartilage, as is shown in Fig. 3. It was obviously a murmur of tricuspid regurgitation, heard most distinctly over the infundibulum.



FIG. 3.

No abnormal symptoms, connected with the respiratory or urinary systems, were present. The patient had some insomnia, followed by restless slumber with alarming dreams, and he had a distinct tremor throughout the entire muscular system.

Under appropriate treatment the nervous disturbances passed away, and the patient was transferred to Ward 22, where he speedily lost all the swelling and breathlessness. The

physical signs connected with the heart had in great part disappeared when he was discharged.

In this case there could be no doubt that the murmur described was due to escape at the right auriculo-ventricular orifice, and its localisation throws much light on the question that has been discussed.

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